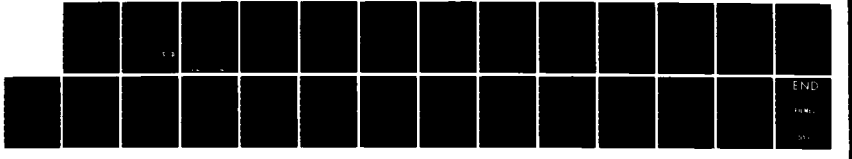
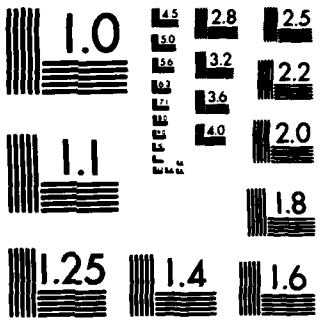
AD-A148 847 FOOD DEPRIVATION AND EXERCISE IN THE HEAT: THERMOREGULATORY AND METABOLIC..(U) ARMY RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MA 1/1

UNCLASSIFIED R P FRANCESCONI ET AL. 05 DEC 84 F/G 6/19 NL





Food Deprivation and Exercise in the Heat: Thermoregulatory and Metabolic Effects

Ralph P. Francesconi, Ph.D.

and

Roger W. Hubbard, Ph.D.

US Army Research Institute of Environmental Medicine

Natick, Massachusetts 01760-5007

Send Proofs To:

Dr. Ralph Francesconi
Heat Research Division
US Army Res. Inst. Environ. Med.
Natick, MA 01760-5007

UNCLAS

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER M 8/85	2. GOVT ACCESSION NO. AD-A148 847	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) Food Deprivation and Exercise in the Heat: Thermoregulatory and Metabolic Effects		5. TYPE OF REPORT & PERIOD COVERED 6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) Ralph P. Francesconi, Ph.D and Roger W. Hubbard, Ph.D		8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS US Army Research Institute of Environmental Medicine; Heat Research Division Natick, MA 01760-5007		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 3M161102BS10 54382101005
11. CONTROLLING OFFICE NAME AND ADDRESS		12. REPORT DATE December 1984 13. NUMBER OF PAGES 22
14. MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office)		15. SECURITY CLASS. (of this report) UNCLAS 15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

DISTRIBUTION A: Approved for public release; distribution is unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Food Deprivation; exercise, glucose; lactate; insulin

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

To determine the effects of food deprivation on the physical, physiological, and metabolic responses to exercise in the heat, adult, male rats (330-360g, N=16/group) were food-drpvived for 24, 48, or 72h. They were then exercised (9.14m/min) in the heat (35.5°C) to hyperthermic exhaustion (Tco 43°C). Food deprivation had no effects on endurance, but ad lib fed controls manifested significantly (P .05) increased Tco and Tsk during the latter portion of the treadmill interval. While plasma osmolality was significantly (P .01) increased in all groups as a result of the heat/exercise contingency, hematocrit

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

UNCLAS

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

ratios were elevated (P .01) as a result of 48 and 72h of food deprivation. Food deprivation resulted in severe hypoglycemia following exercise (P .01), and these decrements were accompanied by marked (P .01) reductions in circulating insulin levels. Prolonged food deprivation (48 and 72h) resulted in significant (P .01) hypertriglyceridemia and hyperlactacidemia subsequent to exercise. Levels of sodium, potassium, urea nitrogen, and creatine phosphokinase were unaffected by the food deprivation intervals. We have concluded from these studies that while several thermoregulatory and metabolic responses to exercise in the heat can be significantly affected by food deprivation, short-term endurance capacity was unaltered.

DISPOSITION FORM

For use of this form, see AR 340-15; the proponent agency is TAGO.

REFERENCE OR OFFICE SYMBOL	SUBJECT
SGRD-UE-HR	Request for Clearance of Technical Paper (USARIEM)

TO Commander, USARIEM FROM C, Heat Rsch Division DATE 28 November 1984 CMT 1

RFrancesconi/seph/4877

1. Reference USARIEM Memo 360-1, I request clearance of attached [X] manuscript, [ ] abstract, [ ] presentation, [ ] technical report, [ ] review article. Report Documentation Page, DD Form 1473 (is)(is not) attached.

Title Food Deprivation and Exercise in the Heat: Thermoregulatory and Metabolic Effects

Author(s) Ralph P. Francesconi, Ph.D and Roger W. Hubbard, Ph.D

Intended for publication in Aviation, Space and Environmental Medicine

Intended for presentation before _____

_____ Location _____ Date _____

2. Budget Project No. 3M161102BS10 Cost Code 54382101005

3. Attached contains no classified material. It meets accepted standards for scientific accuracy and propriety. It contains no potentially sensitive or controversial items.

1 Encl
as

ROGER W. HUBBARD, Ph.D
Chief, Heat Research Division

SGRD UEZ ( )

THRU Chief, Admin Svc Br FROM Commander DATE 5 Dec 84 CMT 2

TO C. HEAT RES DIV

[X] Clearance is granted.

[ ] Clearance is not granted.

[ ] This document must be forwarded to USAMRDC for clearance.

1 Encl
as

BRENDAN E. JOYCE
Colonel, MSC
Commanding

CLEARANCE NO. M8/85

NATICK FL 453
1 Dec 83 EDITION OF 1 NOV 82 IS OBSOLETE.

84 12 21 085

DA FORM AUG 80 2496 PREVIOUS EDITIONS WILL BE USED

Abstract

To determine the effects of food deprivation on the physical, physiological, and metabolic responses to exercise in the heat, adult, male rats (330-360g, N=16/group) were food-deprived for 24, 48, or 72h. They were then exercised (9.14m/min) in the heat (35.5°C) to hyperthermic exhaustion (Tco~43°C). Food deprivation had no effects on endurance, but ad lib fed controls manifested significantly ($P<.05$) increased Tco and Tsk during the latter portion of the treadmill interval. While plasma osmolality was significantly ($P<.01$) increased in all groups as a result of the heat/exercise contingency, hematocrit ratios were elevated ($P<.01$) as a result of 48 and 72h of food deprivation. Food deprivation resulted in severe hypoglycemia following exercise ($P<.01$), and these decrements were accompanied by marked ($P<.01$) reductions in circulating insulin levels. Prolonged food deprivation (48 and 72h) resulted in significant ($P<.01$) hypertriglyceridemia and hyperlactacidemia subsequent to exercise. Levels of sodium, potassium, urea nitrogen, and creatine phosphokinase were unaffected by the food deprivation intervals. We have concluded from these studies that while several thermoregulatory and metabolic responses to exercise in the heat can be significantly affected by food deprivation, short-term endurance capacity was unaltered.

Introduction

Several recent reports have concluded that short-term food deprivation (24h) can increase the endurance capacity of adult rats (2,15) despite nearly depleted liver glycogen stores and reduced plasma glucose levels in the food-deprived animals. It has been hypothesized that increased rates of fatty acid oxidation serve to spare muscle glycogen in the fasted animals thus increasing endurance. Dohm et al.(2) quantitated the glycogen levels of exercising muscles in fasted-exhausted rats and in fed-exhausted rats and noted significantly elevated levels in the former thus indicating a muscle glycogen-sparing effect of the previous food deprivation. The impetus for investigating the effects of dietary restriction or manipulation on endurance came from the earlier work of Bergstrom et al.(1) who demonstrated that increasing the glycogen concentration of the working muscle by carbohydrate loading was effective in increasing endurance. Thus, moderate glycogen depletion and repletion have both been associated with increased physical performance.

Using our exercising, heat-stressed rat model of human heat injury (13,14), we have demonstrated that prior pharmacological manipulation of core temperature can likewise affect endurance during work in the heat. For example, pre-induced hypothermia can delay the onset of hyperthermic exhaustion thus prolonging

treadmill endurance in the heat (5,6). Alternatively, we also reported (10) that hyperthermia, induced in rats by central administration of prostaglandin E_1, reduced endurance in the heat. It has also been reported that pharmacologically induced hypothermia is more severe when the test population has been food-deprived for various time intervals (7,11), thus indicating that reduced oxidative fuel supply may be manifested in lowered core temperatures (Tco). Therefore, since acute food deprivation has been associated with elevated physical work performance as well as reduced Tco, it was of interest to us to determine the effects of food deprivation on the subsequent ability to work in the heat. To this end groups of rats were food-deprived for fixed intervals, exercised in the heat to hyperthermic exhaustion, and physiological, thermoregulatory, and metabolic responses were monitored.

Methods

Adult, male rats (250-275g) were purchased from the Charles River Breeding Laboratories (CD1, Wilmington, MA), placed singly in wire-bottomed cages, and held in windowless rooms (21-22^{o}C) with automatically timed fluorescent lighting (on, 0600-1800h). Rats had free access to a complete rodent diet (Ralston-Purina, St. Louis, MO) and fresh water. Weights at the time of food

deprivation were selected (330-360g) to achieve maximal consistency in initial run weights at the time of the experimental trials. Experimental rats (N=16/group) were food-deprived for 24, 48, or 72h while a control group (CONT) had continuous access to the nutritionally complete diet. At the time of the experimental run CONT rats had a mean body weight of 322.7 $\pm$ 4.5g, ($\bar{X} \pm SE_x$). The group food-deprived for 24h (24FD) had a mean body weight of 297.3 $\pm$ 3.7g; 48FD and 72FD groups weighed 290.9 $\pm$ 7.1g and 301.4 $\pm$ 4.0g, respectively, immediately prior to the start of the exercise/heat contingency. Food and water consumption and body weight changes were carefully monitored prior to and during the food deprivation intervals.

On the day before an experimental run each animal was fitted with a permanently implanted venous catheter (Silastic, external jugular vein) for rapid and convenient blood sampling. On the following day, just prior to the heat/treadmill contingency, a blood sample (0.8ml) was taken, hematocrit ratios were immediately measured, and the blood sample was centrifuged (10,000g, 4^{o}C). Osmolality (Precision Systems, Inc., Sudbury, MA) was quantitated on an aliquot of the fresh plasma sample, and the remainder was deep-frozen (-20^{o}C) for subsequent analysis.

The animals were then removed to a large (3x4x2m) stainless steel chamber maintained at 35.5^{o}C (25-35%rh) and exercised (9.14m/min) to hyperthermic exhaustion (T_{co} ~ 43^{o}C). During the experimental run core (Tco, thermistor inserted to a depth of

6cm) and tail-skin (Tsk, midlength on the tail) were measured on a minute-by-minute basis. Immediately after termination of the treadmill run (Tco = 43°C, animal unable to right itself), a second blood sample was taken and treated identically as the first.

Both plasma samples were analyzed for several indices of heat/exercise injury as well as carbohydrate and lipid metabolism. Circulating insulin levels were assayed using commercially available radioimmunoassay test kits produced by Serono Labs, Inc. (Randolph, MA) by procedures described in their technical bulletin. Potassium (K^+) and sodium (Na^+) were quantitated by standard flame photometric techniques (Radiometer, Copenhagen) while lactate was measured by commercially available test kits and procedures (Sigma Chem. Co., St. Louis, MO). Triglycerides, α-hydroxybutyrate dehydrogenase (HBDH), glucose, urea nitrogen (UN), and creatine phosphokinase (CPK) were all measured with commercially prepared test kits and specified procedures (Gilford Diagnostics, Cleveland, OH). All assays were performed on a semi-automated Gilford spectrophotometer (Stasar IV) and read at 340 nm except the triglycerides which were quantitated at 500 nm.

Statistical analysis was performed by analysis of variance (18) followed by the application of Tukey's t test corrected for multiple comparisons (17). For comparison of Tco there were several instances where unequal N remained in the various groups

due to attainment of hyperthermic exhaustion; in these cases Dunnett's t test, corrected for several comparisons, was used to determine statistical significance (17). The null hypothesis was rejected at $P<.05$.

Results

Fig. 1 illustrates the mean Tco response to exercise in the heat to hyperthermic exhaustion of the CONT and experimental groups. The results demonstrated that food deprivation for up to 72h had no significant effects on Tco. As treadmill time progressed, however, there developed an exacerbated hyperthermia among the CONT rats. Despite no significant differences in mean Tco at 20 min, by 25 and 26 min Tco among controls was significantly ($P<.05$) higher than either the 48 FD or 72 FD. After 27 min Tco of the control group was significantly ($P<.05$) higher than all three experimental groups, and these differences persisted through 30 min. Mean Tsk responses, depicted in Fig. 2, manifested rather analogous responses. For example, after 5 min of exercise in the heat no significant differences (P=NS) among groups were noted in mean Tsk. However, after 10 min, Tsk CONT was significantly ($P<.05$) greater than that of 72FD, but not 24 or 48 FD. However, by 20 min treadmill time Tsk CONT was significantly ($P<.05$) elevated when compared with all 3 experimental groups.

Table 1 demonstrates slight trends toward increased levels of CPK following exercise in the heat to hyperthermic exhaustion; actually, significance was not attained due to the wide variability characteristic of these brief endurances at the slow speed selected. All 4 groups manifested significant ($P<.01$) increments in plasma osmolality subsequent to the heat/exercise regimen. Hematocrit levels were, for each group, unaffected by exercise in the heat; however, increasing intervals of food deprivation were characterized by increasing hematocrit levels such that mean hematocrit at 48 FD and 72 FD were significantly ($P<.01$) elevated when compared to either CONT or 24 FD.

Glucose levels (Table 2) were generally decreased ($P<.05$) prior to exercise in the heat as a result of 24-72h of food deprivation. Following exercise in the heat, there were no changes in glucose levels in the CONT group, but all three FD groups manifested severe hypoglycemia ($P<.01$). Insulin concentrations generally mirrored closely circulating glucose levels. Thus, food deprivation resulted in significant ($P<.01$) decrements in circulating insulin in all three groups. While exercise in the heat further depressed these already low levels, no signficant changes arose due to the heat/exercise regimen. While plasma triglycerides were significantly ($P<.05$) reduced by exercise in the heat in the CONT and 24 FD groups, more prolonged food deprivation was associated with increased levels of triglycerides following completion of the exercise regimen. In

fact, following 48 and 72h of food deprivation triglyceride levels were slightly elevated in the post-run vs. the pre-run samples. Hydroxybutyrate dehydrogenase was generally increased as a result of the exercise/heat regimen, and at 48 FD, the post-run level was significantly ($p<.05$) elevated when compared to the CONT post-run indicating an effect of food deprivation also.

Table 3 illustrates that Na^+ levels were generally unaffected by either exercise in the heat to hyperthermic exhaustion or the food deprivation regimen. K^+ ($P<.05$) and urea nitrogen ($P<.01$) levels were significantly increased by exercise in the heat, but not affected by food deprivation. It is noteworthy that lactate levels were significantly ($P<.01$) increased in all groups by the exercise/heat stress, and, also, that the increments were exaggerated by prolonged food deprivation. For example, post-run levels at 48 FD and 72 FD were significantly ($P<.01$) increased when compared to the post-run levels recorded at 24 FD or in the CONT group.

DISCUSSION

The current experimental protocol did not affect endurance in the heat; the intervals required to reach hyperthermic exhaustion ranged from a low of 30.9 min (48 FD) to a high of 35.1 min (24 FD). Using separate groups of rats we determined that food deprivation for 24-72h drastically reduced the mean

glycogen content of the liver to approximately 1-3% of control levels. Thus, under conditions of the present experiment, it is clear that endurance was not related to initial liver glycogen content. Further, despite no differences in the time to hyperthermic exhaustion, circulating glucose levels were markedly reduced in the food-deprived animals. Thus, these results are consistent with those of Dohn et al.(2) who concluded that blood glucose level is probably not a limiting factor in exhaustion.

To the best of our knowledge, the relationship between food intake, restriction, and thermoregulatory responses during exercise in the heat had not been investigated previously. Despite no changes in initial Tco after 72h of food deprivation, the present results indicate that heat gain during exercise in the heat was decreased as a result of prior food deprivation. It had been previously reported (4) that restricted food intake was accompanied by a decreased metabolic rate, but the effects of heat and exercise were not reported. There appears to be no physiological benefit to this decrease in heat gain, however, since endurance in the heat was unaffected. In an earlier study McCormick et al.(19) had demonstrated that food deprivation increased the survivability of chicks when they were exposed to high ambient temperatures; however, core temperatures were not reported in this study.

The responses of tail-skin temperature to exercise in the heat indicated that the increased heat gain in the CONT group was

probably related more to metabolic alterations than to heat dissipation since Tsk was generally higher in this group. The reduced mean Tsk in the FD groups may be related to the increased hematocrits and apparently decreased plasma volumes in these animals. Generally, rats are regarded as prandial drinkers and food deprivation usually reduces water consumption. Indeed, in the current experiments food deprivation resulted in decrements in water consumption ranging from 60-80%. During exercise in the heat, CONT rats manifested a mean weight (water) loss of 8.9g. The comparable values for the 24, 48, and 72 FD groups were 7.5, 6.9, and 5.6g, respectively. Thus, the thermoregulatory and weight change data are consistent with the hypothesis that there occurred an increased metabolic heat production in the CONT group together with a decreased peripheral blood flow in the FD groups secondary to reduced fluid consumption and plasma volume. Additional studies on the themoregulatory effects of food deprivation during exercise in the heat are indicated.

The intensity of exercise used in these experiments was relatively mild; thus, it is not unexpected that glucose uptake by the exercising muscle (3) did not exceed the ability of the liver to regenerate endogenous supplies (16). Hence, in the CONT group circulating glucose was unaffected by the exercise regimen. However, in the FD groups initial hepatic glycogen depletion resulted in significant decrements in plasma glucose prior to exercise with further decreases in the post-exercise samples.

These exacerbated decreases following exercise in the FD groups were closely associated with extremely depressed insulin levels in these samples. While plasma insulin concentration is ordinarily decreased during prolonged exercise (3), the present results demonstrated that this short-term exercise protocol had no effects on insulin levels when adequate food was ingested.

It is noteworthy that while triglycerides were decreased following exercise in the heat in the CONT and 24 FD groups, more prolonged food deprivation was associated with slight elevations in triglycerides subsequent to exercise. These observations apparently indicate that at the more prolonged intervals of food deprivation, the exercising rats were dependent upon mobilization and oxidation of body fat stores to supply the substrate for metabolic energy production. This extensive mobilization was apparently reflected in the increased triglyceride levels noted after exercise in the heat. Analogously, in the 48 and 72 FD groups, plasma lactate levels also manifested exaggerated elevations following exercise. We had previously demonstrated that both alcohol consumption (9) and chronic chlorpromazine administration (8) were associated with a hyperlactacidemia following exercise in the heat. However, to the best of our knowledge the effects of food deprivation on heat (12) or exercise (20) induced lactacidemia have not been reported.

We have concluded from these studies that food deprivation may affect thermoregulatory responses to exercise in the heat;

however, endurance capacity was not affected by prior food deprivation for up to 72h. Hematocrit levels increased significantly with more prolonged food deprivation. Following exercise in the heat circulating glucose levels were severely depressed in the food-deprived animals as were plasma insulin levels. However, in the post-exercise blood samples of the 48 FD and 72 FD groups, plasma levels of triglycerides and lactate were significantly increased when compared with post-run levels in the CONT and 24 FD groups. Thus, despite no noteable effects of food deprivation on physical performance in the heat, several marked metabolic and thermoregulatory effects were observed, particularly after more prolonged food deprivation.

Figure Legend

Fig. 1 illustrates the effects of food deprivation on the Tco response to exercise (9.14m/min) in the heat (35.5°C) to hyperthermic exhaustion (Tco = 43°). Mean values are depicted for an N of 16 in each group. Standard errors of the mean are not depicted because in many instances these fell within the range of the symbols.

Fig. 2 demonstrates the effects of food deprivation on the Tsk responses to exercise in the heat. All conditions are as noted under Fig.1 Tail-skin temperatures were recorded mid-length on the tail.

Acknowledgements

The authors gratefully acknowledge the skilled technical assistance of Natalie Leva, Virginia Pease and Jeffery Young. We express our gratitude to Susan Henry and Diane Danielski for their technical assistance in preparing the manuscript.

The views, opinions, and findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation. In conducting the research described in this report, the investigators adhered to the "Guide for the Care and Use of Laboratory Animals," as prepared by the committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council.

References

1. Bergstrom, J., L. Hermansen, E. Hultman, and B. Saltin. Diet, muscle glycogen and physical performance. Acta Physiol. Scand. 71:140-150, 1967.

2. Dohm, G.L., E.B. Tapscott, H.A. Barakat, and G.J. Kasperek. Influence of fasting on glycogen depletion in rats during exercise. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55:830-833, 1983.

3. Felig, P. and J. Wahren. Fuel homeostasis in exercise. New Engl. J. Med. 293:1078-1084, 1975.

4. Forsum, E., P. E. Hillman, and M.C. Nesheim. Effect of energy restriction on total heat production, basal metabolic rate, and specific dynamic action of food in rats. J. Nutr. 111:1691-1697, 1981.

5. Francesconi, R. and M. Mager. Hypothermia induced by chlorpromazine or L-tryptophan: effect on treadmill performance in the heat. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 47:813-817, 1979.

6. Francesconi, R. and M. Mager. Hypothermia induced by 5-thio-D-glucose: effects on treadmill performance in the heat. Aviat. Space Environ. Med. 51:754-758, 1980.

7. Francesconi, R. and M. Mager. 5-Thio-D-glucose: hypothermic responses in mice. Am. J. Physiol. 239 (Reg. Integ. Comp. Physiol. 8): R214-R218, 1980.

8. Francesconi, R. and M. Mager. Chronic chlorpromazine administration in rats: effects on ability to work in the heat. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 50:509-512, 1981.

9. Francesconi, R. and M. Mager. Alcohol consumption in rats: effects on work capacity in the heat. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 50:1006-1010, 1981.

10. Francesconi, R. and M. Mager. Prostagladin E, hyperthermia: effects on ability to work in the heat. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 51:62-67, 1981.

11. Freinkel, N., B.E. Metzger, E. Harris, S. Robinson, and M. Mager. The hypothermia of hypoglycemia. New Engl. J. Med. 287:841-845, 1972.

12. Harrison, M.H. Effects of acute heat stress on tissue carbohydrate in fasted rats. Aviat. Space Environ. Med. 47:165-167, 1976.

13. Hubbard, R. W., W. D. Bowers, and M. Mager. A study of physiological, pathological and biochemical changes in rats with heat- and/or work-induced disorders. Isr. J. Med. Sci. 12:884-886, 1976.

14. Hubbard, R.W., W.T. Matthew, J.D. Linduska, F.C. Curtis, W.D. Bowers, I. Leav, and M. Mager. The laboratory rat as a model for hyperthermic syndromes in humans. Am.J. Physiol. 231:1119-1123, 1976.

15. Kasperek, G.J., G.L. Dohm, E.B. Tapscott, and T. Powell. Effect of exercise on liver protein loss and lysosomal enzyme levels in fed and fasted rats. Proc. Soc. Exp. Biol. Med. 164:430-434, 1980.

16. Koivisto, V.A., S-L. Karonen, and E.A. Nikkila. Carbohydrate ingestion before exercise: comparison of glucose, fructose, and sweet placebo. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 51:783-787, 1981.

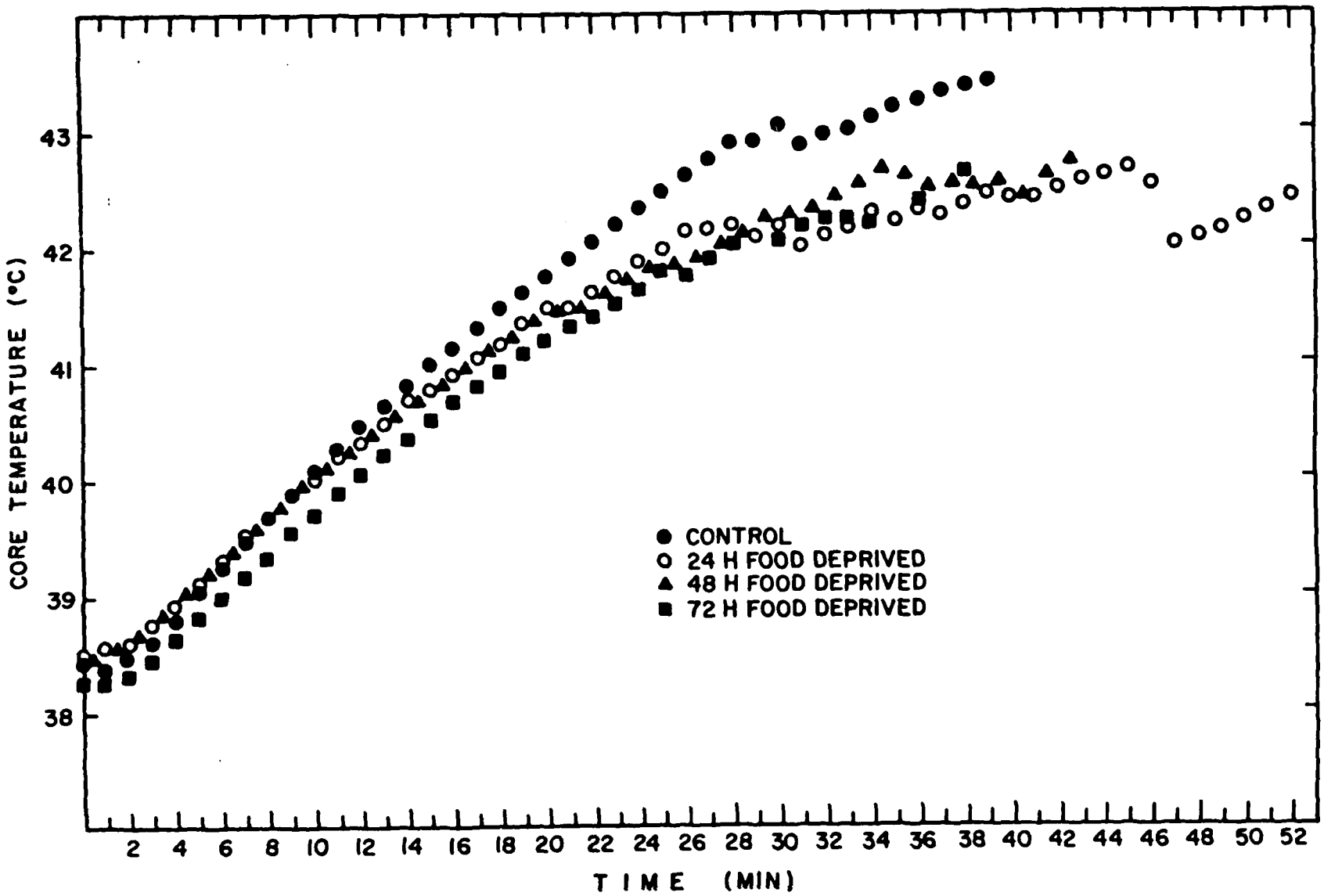
17. Li, C.C. Introduction to Experimental Statistics. New York, N.Y.: McGraw-Hill, 1964, p425.

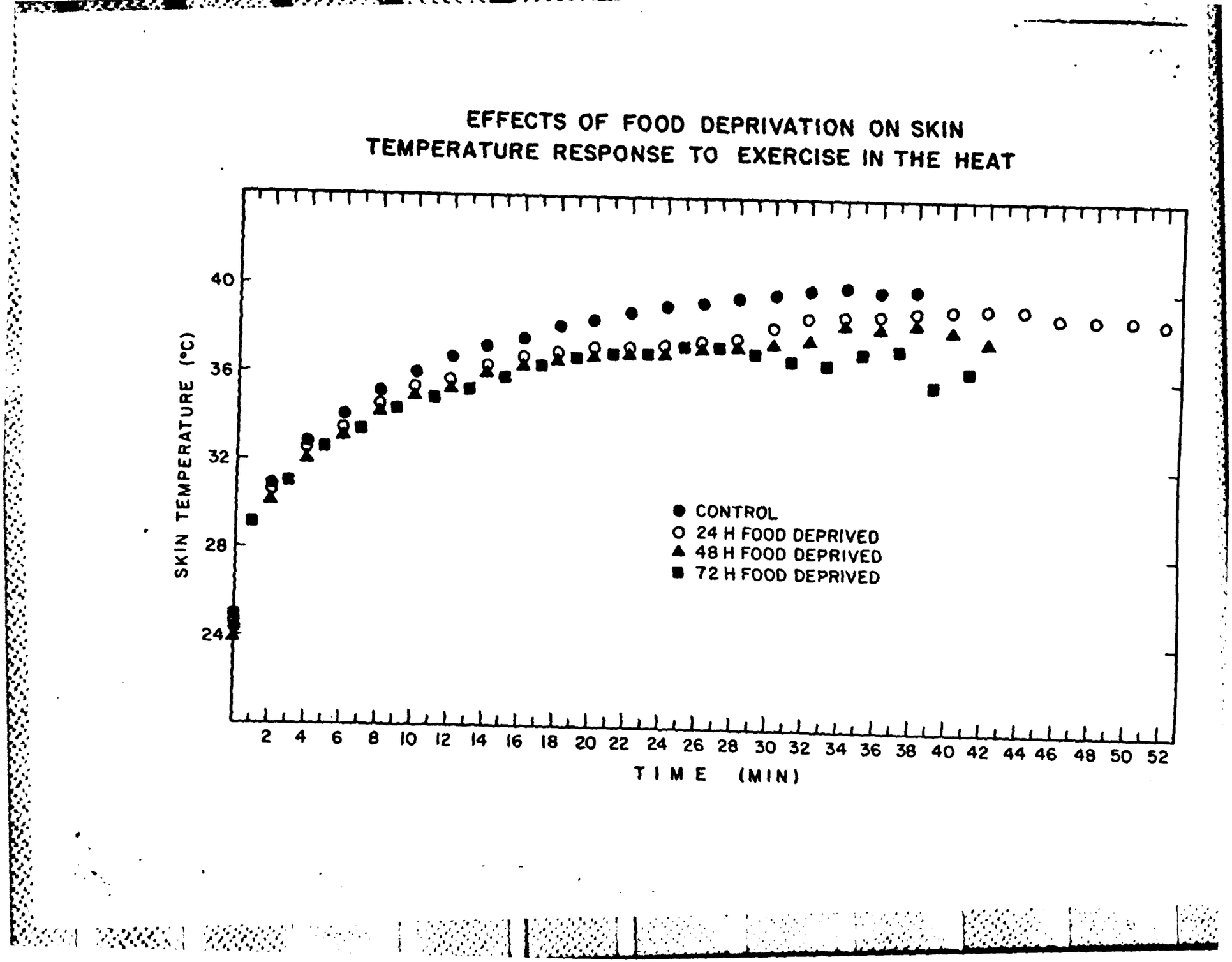
18. Lindquist, E. Design and Analysis of Experiments in Psychology and Education. Boston, MA: Houghton-Mifflin, 1953, p56, 269.

19. McCormick, C.C., J. D. Garlich, and F.W. Edens. Fasting and diet affect the tolerance of young chickens exposed to acute heat stress. J. Nutr. 109:1797-1809, 1979.

20. Saltin, B. and L. Hermansen. Glycogen stores and prolonged severe exercise. Symp. Swedish Nutr. Fdn. V: 32-46, 1967.

EFFECTS OF FOOD DEPRIVATION ON CORE TEMPERATURE RESPONSE TO EXERCISE IN THE HEAT




EFFECTS OF FOOD DEPRIVATION ON SKIN
TEMPERATURE RESPONSE TO EXERCISE IN THE HEAT
SKIN TEMPERATURE (°C)
40
36
32
28
24
CONTROL
24 H FOOD DEPRIVED
48 H FOOD DEPRIVED
72 H FOOD DEPRIVED
2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40 42 44 46 48 50 52
TIME (MIN)

EFFECTS OF FOOD DEPRIVATION ON CPK, OSMOLALITY, AND HEMATOCRIT PRIOR AND SUBSEQUENT TO EXERCISE IN THE HEAT

	CONTROL		24 HOUR FOOD DEPRIVATION		48 HOUR FOOD DEPRIVATION		72 HOUR FOOD DEPRIVATION	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
CREATINE PHOSPHOKINASE (U/L)	68.2 ±11.7	104.0 ±15.4	81.0 ±6.9	124.7 ±35.2	63.0 ±4.9	237.2 ±84.0	64.8 ±11.7	162.2 ±54.1
OSMOLALITY (MOSM/KG)	295.0 ±1.1	307.1 ±1.4	292.8 ±1.2	302.9 ±1.0	292.4 ±0.8	308.3 ±1.3	295.9 ±0.8	312.4 ±1.7
HEMATOCRIT (% RBC)	44.3 ±0.9	43.1 ±0.8	46.7 ±0.7	45.4 ±0.8	50.9 ±0.6	52.0 ±1.2	51.9 ±0.6	53.8 ±0.8

EFFECTS OF FOOD DEPRIVATION ON INDICES OF CARBOHYDRATE AND LIPID METABOLISM PRIOR AND SUBSEQUENT TO EXERCISE IN THE HEAT

	CONTROL		24 HOUR FOOD DEPRIVATION		48 HOUR FOOD DEPRIVATION		72 HOUR FOOD DEPRIVATION	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
GLUCOSE (MG/DL)	147.5 ±3.3	135.5 ±6.9	112.1 ±2.5	76.1 ±5.0	119.4 ±4.8	69.1 ±7.9	117.2 ±2.9	69.3 ±5.1
INSULIN (μU/ML)	23.2 ±2.0	24.9 ±3.8	9.4 ±0.9	5.7 ±0.3	8.1 ±0.6	5.4 ±0.5	9.1 ±0.9	5.1 ±0.3
TRIGLYCERIDES (MG/DL)	32.2 ±3.0	16.2 ±1.6	43.5 ±3.8	30.1 ±1.7	39.3 ±3.7	46.2 ±4.2	37.2 ±2.3	41.6 ±4.2
HYDROXYBUTYRATE DEHYDROGENASE (U/L)	91.4 ±9.6	193.8 ±15.0	109.7 ±19.3	251.4 ±41.7	114.5 ±17.8	386.5 ±94.0	129.8 ±14.9	314.7 ±27.1

EFFECTS OF FOOD DEPRIVATION ON INDICES OF HEAT/EXERCISE INJURY PRIOR AND SUBSEQUENT TO EXERCISE IN THE HEAT

	CONTROL		24 HOUR FOOD DEPRIVATION		48 HOUR FOOD DEPRIVATION		72 HOUR FOOD DEPRIVATION	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
SODIUM (MEQ/L)	148.5 ±0.9	153.6 ±1.3	148.1 ±1.5	151.1 ±1.7	144.4 ±0.9	146.3 ±1.6	145.9 ±0.6	150.9 ±0.6
POTASSIUM (MEQ/L)	4.86 ±0.13	6.18 ±0.21	4.71 ±0.15	5.24 ±0.29	4.52 ±0.14	5.93 ±0.40	4.66 ±0.37	6.23 ±0.40
UREA NITROGEN (MG/DL)	15.7 ±0.6	23.0 ±0.8	15.4 ±0.7	27.5 ±2.2	15.5 ±0.8	25.4 ±1.2	14.5 ±0.9	24.9 ±0.9
LACTATE (MG/DL)	22.6 ±2.2	68.6 ±7.8	22.4 ±2.6	58.3 ±5.5	28.3 ±2.4	102.1 ±6.8	35.9 ±5.3	119.9 ±5.7

END

FILMED

1-85

DTIC